FILE 'HOME' ENTERED AT 12:08:53 ON 14 AUG 2002

=> file medline biosis caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 12:09:07 ON 14 AUG 2002

FILE 'BIOSIS' ENTERED AT 12:09:07 ON 14 AUG 2002

FILE 'CAPLUS' ENTERED AT 12:09:07 ON 14 AUG 2002

=> s psoralen and site (w) direct? (3a) Mutagen?
L1 25 PSORALEN AND SITE (W) DIRECT? (3A) MUTAGEN?

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2 19 DUP REM L1 (6 DUPLICATES REMOVED)

=> d 1-19 ti

L2 ANSWER 1 OF 19 MEDLINE
TI Human XPA and RPA DNA repair proteins participate in specific recognition of triplex-induced helical distortions.

L2 ANSWER 2 OF 19 MEDLINE DUPLICATE 1
TI Chromosome targeting at short polypurine sites by cationic triplex-forming oligonucleotides.

L2 ANSWER 3 OF 19 MEDLINE
TI Stability of DNA triplexes on shuttle vector plasmids in the replication pool in mammalian cells.

L2 ANSWER 4 OF 19 MEDLINE
TI Activation of human gamma-globin gene expression via triplex-forming oligonucleotide (TFO)-directed mutations in the gamma-globin gene 5' flanking region.

L2 ANSWER 5 OF 19 MEDLINE
TI Triplex formation by oligonucleotides containing 5-(1-propynyl)-2'-deoxyuridine: decreased magnesium dependence and improved intracellular gene targeting.

L2 ANSWER 6 OF 19 MEDLINE
TI Mutagenesis mediated by triple helix-forming oligonucleotides conjugated to **psoralen**: effects of linker arm length and sequence context.

L2 ANSWER 7 OF 19 CAPLUS COPYRIGHT 2002 ACS
TI Gene targeting via triple helix formation

L2 ANSWER 8 OF 19 MEDLINE
TI Processing of targeted **psoralen** cross-links in Xenopus oocytes.

L2 ANSWER 9 OF 19 CAPLUS COPYRIGHT 2002 ACS
TI Chemically modified triplex-forming oligonucleotide for **site-directed mutagenesis**

L2 ANSWER 10 OF 19 MEDLINE
TI Mutagenesis by third-strand-directed **psoralen** adducts in repair-deficient human cells: high frequency and altered spectrum in a xeroderma pigmentosum variant.

L2 ANSWER 11 OF 19 CAPLUS COPYRIGHT 2002 ACS
TI Chemically modified oligonucleotide for **site-directed mutagenesis**

L2 ANSWER 12 OF 19 MEDLINE
TI Targeted mutagenesis in mammalian cells mediated by intracellular triple helix formation.

L2 ANSWER 13 OF 19 MEDLINE DUPLICATE 2
TI Structural changes in base-paired region 28 in 16 S rRNA close to the decoding region of the 30 S ribosomal subunit are correlated to changes in tRNA binding.

L2 ANSWER 14 OF 19 MEDLINE
TI Site-specific targeting of **psoralen** photoadducts with a triple helix-forming oligonucleotide: characterization of **psoralen** monoadduct and crosslink formation.

L2 ANSWER 15 OF 19 MEDLINE
TI Targeted mutagenesis of DNA using triple helix-forming oligonucleotides linked to **psoralen**.

L2 ANSWER 16 OF 19 MEDLINE
TI Targeted mutagenesis of simian virus 40 DNA mediated by a triple helix-forming oligonucleotide.

L2 ANSWER 17 OF 19 MEDLINE
TI Inhibition of gene expression by triple helix-directed DNA cross-linking at specific sites.

L2 ANSWER 18 OF 19 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI SEQUENCE-SPECIFIC PHOTO-INDUCED CROSS-LINKING OF THE TWO STRANDS OF DOUBLE-HELICAL DNA BY A **PSORALEN** COVALENTLY LINKED TO A TRIPLE HELIX-FORMING OLIGONUCLEOTIDE.

L2 ANSWER 19 OF 19 MEDLINE DUPLICATE 3
TI Protein L18 binds primarily at the junctions of helix II and internal loops A and B in Escherichia coli 5 S RNA. Implications for 5 S RNA structure.

=> d 3, 8 bib ab

L2 ANSWER 3 OF 19 MEDLINE
AN 2001105971 MEDLINE
DN 20564272 PubMed ID: 10993885
TI Stability of DNA triplexes on shuttle vector plasmids in the replication pool in mammalian cells.
AU Lin F L; Majumdar A; Klotz L C; Reszka A P; Neidle S; Seidman M M
CS Laboratory of Molecular Genetics, National Institute on Aging, National Institutes of Health, Baltimore, Maryland 21224, USA.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Dec 15) 275 (50) 39117-24. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 200102
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208
AB Triple helix-forming oligonucleotides may be useful as gene-targeting reagents in vivo, for applications such as gene knockout. One important property of these complexes is their often remarkable stability, as demonstrated in solution and in cells following transfection. Although encouraging, these measurements do not necessarily report triplex stability in cellular compartments that support DNA functions such as replication and mutagenesis. We have devised a shuttle vector plasmid assay that reports the stability of triplexes on DNA that undergoes replication and mutagenesis. The assay is based on plasmids with novel variant supF tRNA genes containing embedded sequences for triplex formation and **psoralen** cross-linking. Triple helix-forming oligonucleotides were linked to **psoralen** and used to form triplexes on the plasmids. At various times after introduction into cells, the **psoralen** was activated by exposure to long wave ultraviolet light (UVA). After time for replication and mutagenesis, progeny plasmids were recovered and the frequency of plasmids with mutations in the supF gene determined. Site-specific mutagenesis by **psoralen** cross-links was dependent on precise placement of the **psoralen** by the triple helix-forming oligonucleotide at the time of UVA treatment. The results indicated that both pyrimidine and purine motif triplexes were much less stable on replicated DNA than on DNA in vitro or in total transfected DNA. Incubation of cells with amidoanthraquinone-based triplex stabilizing compounds enhanced the stability of the pyrimidine triplex.

L2 ANSWER 8 OF 19 MEDLINE
AN 1998001593 MEDLINE
DN 98001593 PubMed ID: 9343428
TI Processing of targeted **psoralen** cross-links in Xenopus oocytes.
AU Segal D J; Faruqi A F; Glazer P M; Carroll D
CS Department of Biochemistry, University of Utah School of Medicine, Salt Lake City 84132, USA.
NC CA64186 (NCI)
GM50739 (NIGMS)
SO MOLECULAR AND CELLULAR BIOLOGY, (1997 Nov) 17 (11) 6645-52.
Journal code: 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971121
AB **Psoralen** cross-links have been shown to be both mutagenic and recombinagenic in bacterial, yeast, and mammalian cells. Double-strand breaks (DSBs) have been implicated as intermediates in the removal of **psoralen** cross-links. Recent work has suggested that site-specific mutagenesis and recombination might be achieved through the use of targeted **psoralen** adducts. The fate of plasmids containing **psoralen** adducts was evaluated in Xenopus oocytes, an experimental system that has well-characterized recombination capabilities and advantages in the analysis of intermediates in DNA metabolism. **Psoralen** adducts were delivered to a specific site by a triplex-forming oligonucleotide. These lesions are clearly recognized and processed in oocytes, since mutagenesis was observed at the target site. The spectrum of induced mutations was compared with that found in similar studies in mammalian cells. Plasmids carrying multiple random adducts were preferentially degraded, perhaps due to the introduction of DSBs. However,

when DNAs carrying site-specific adducts were examined, no plasmid loss was observed and removal of cross-links was found to be very slow. Sensitive assays for DSB-dependent homologous recombination were performed with substrates with one or two cross-link sites. No adduct-stimulated recombination was observed with a single lesion, and only very low levels were observed with paired lesions, even when a large proportion of the cross-links was removed by the oocytes. We conclude that DSBs or other recombinagenic structures are not efficiently formed at **psoralen** adducts in Xenopus oocytes. While **psoralen** is not a promising reagent for stimulating site-specific recombination, it is effective in inducing targeted mutations.

=> d 1, 2 bib ab

L2 ANSWER 1 OF 19 MEDLINE
AN 2002263266 MEDLINE
DN 21980606 PubMed ID: 11972036
TI Human XPA and RPA DNA repair proteins participate in specific recognition of triplex-induced helical distortions.
AU Vasquez Karen M; Christensen Jesper; Li Lei; Finch Rick A; Glazer Peter M
CS Department of Carcinogenesis, University of Texas M. D. Anderson Cancer Center, Science Park-Research Division, Park Road 1-C, Smithville, TX 78957, USA.. kvasquez@sprd1.mdacc.tmc.edu
NC AI26109 (NIAID)
CA64186 (NCI)
CA75723 (NCI)
CA93729 (NCI)
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Apr 30) 99 (9) 5848-53.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200206
ED Entered STN: 20020511
Last Updated on STN: 20020612
Entered Medline: 20020611
AB Nucleotide excision repair (NER) plays a central role in maintaining genomic integrity by detecting and repairing a wide variety of DNA lesions. Xeroderma pigmentosum complementation group A protein (XPA) is an essential component of the repair machinery, and it is thought to be involved in the initial step as a DNA damage recognition and/or confirmation factor. Human replication protein A (RPA) and XPA have been reported to interact to form a DNA damage recognition complex with greater specificity for damaged DNA than XPA alone. The mechanism by which these two proteins recognize such a wide array of structures resulting from different types of DNA damage is not known. One possibility is that they recognize a common feature of the lesions, such as distortions of the helical backbone. We have tested this idea by determining whether human XPA and RPA proteins can recognize the helical distortions induced by a DNA triple helix, a noncanonical DNA structure that has been shown to induce DNA repair, mutagenesis, and recombination. We measured binding of XPA and RPA, together or separately, to substrates containing triplexes with three, two, or no strands covalently linked by **psoralen** conjugation and photoaddition. We found that RPA alone recognizes all covalent triplex structures, but also forms multivalent nonspecific DNA aggregates at higher concentrations. XPA by itself does not recognize the substrates, but it binds them in the presence of RPA. Addition of XPA decreases the nonspecific DNA aggregate formation. These results support the hypothesis that the NER machinery is targeted to helical distortions

and demonstrate that RPA can recognize damaged DNA even without XPA.

L2 ANSWER 2 OF 19 MEDLINE DUPLICATE 1
AN 2001553860 MEDLINE
DN 21486479 PubMed ID: 11504712
TI Chromosome targeting at short polypurine sites by cationic triplex-forming oligonucleotides.
AU Vasquez K M; Dagle J M; Weeks D L; Glazer P M
CS Department of Therapeutic Radiology and Genetics, Yale University School of Medicine, New Haven, Connecticut 06520-8040, USA.
NC CA64186 (NCI)
CA75723 (NCI)
GM/OD56277 (NIGMS)
GM54791 (NIGMS)
HD27748 (NICHD)
HL62178 (NHLBI)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 Oct 19) 276 (42) 38536-41.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20011016
Last Updated on STN: 20020122
Entered Medline: 20011204
AB Triplex-forming oligonucleotides (TFOs) bind specifically to duplex DNA and provide a strategy for site-directed modification of genomic DNA. Recently we demonstrated TFO-mediated targeted gene knockout following systemic administration in animals. However, a limitation to this approach is the requirement for a polypurine tract (typically 15-30 base pairs (bp)) in the target DNA to afford high affinity third strand binding, thus restricting the number of sites available for effective targeting. To overcome this limitation, we have investigated the ability of chemically modified TFOs to target a short (10 bp) site in a chromosomal locus in mouse cells and induce site-specific mutations. We report that replacement of the phosphodiester backbone with cationic phosphoramidate linkages, either N,N-diethylethylenediamine or N,N-dimethylaminopropylamine, in a 10-nucleotide, **psoralen**-conjugated TFO confers substantial increases in binding affinity in vitro and is required to achieve targeted modification of a chromosomal reporter gene in mammalian cells. The triplex-directed, site-specific induction of mutagenesis in the chromosomal target was charge- and modification-dependent, with the activity of N,N-diethylethylenediamine > N,N-dimethylaminopropylamine phosphodiester, resulting in 10-, 6-, and <2-fold induction of target gene mutagenesis, respectively. Similarly, N,N-diethylethylenediamine and N,N-dimethylaminopropylamine TFOs were found to enhance targeting at a 16-bp G:C bp-rich target site in a chromatinized episomal target in monkey COS cells, although this longer site was also targetable by a phosphodiester TFO. These results indicate that replacement of phosphodiester bonds with positively charged N,N-diethylethylenediamine linkages enhances intracellular activity and allows targeting of relatively short polypurine sites, thereby substantially expanding the number of potential triplex target sites in the genome.

=> d his

(FILE 'HOME' ENTERED AT 12:08:53 ON 14 AUG 2002)

FILE 'MEDLINE, BIOSIS, CAPLUS' ENTERED AT 12:09:07 ON 14 AUG 2002
L1 25 S PSORALEN AND SITE (W) DIRECT? (3A) MUTAGEN?

L2 19 DUP REM L1 (6 DUPLICATES REMOVED)

=> d 6 14 bib ab

L2 ANSWER 6 OF 19 MEDLINE
AN 1998184132 MEDLINE
DN 98184132 PubMed ID: 9523530
TI Mutagenesis mediated by triple helix-forming oligonucleotides conjugated to **psoralen**: effects of linker arm length and sequence context.
AU Raha M; Lacroix L; Glazer P M
CS Department of Therapeutic Radiology, Yale University School of Medicine, New Haven, CT 06520-8040, USA.
NC CA64186 (NCI)
P30AR41942 (NIAMS)
SO PHOTOCHEMISTRY AND PHOTOBIOLOGY, (1998 Mar) 67 (3) 289-94.
Journal code: 0376425. ISSN: 0031-8655.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199805
ED Entered STN: 19980520
Last Updated on STN: 19980520
Entered Medline: 19980513
AB Targeted mutagenesis and gene knock-out can be mediated by triple helix-forming oligonucleotides (TFO) linked to mutagenic agents, such as **psoralen**. However, this strategy is limited by the availability of homopurine/homopyrimidine stretches at or near the target site because such sequences are required for high-affinity triplex formation. To overcome this limitation, we have tested TFO conjugated to **psoralen** via linker arms of lengths varying from 2 to 86 bonds, thereby designed to deliver the **psoralen** at varying distances from the third strand binding site present at the 3' end of the supFG1 mutation reporter gene. Following triplex formation and UVA irradiation, mutations were detected using an SV40-based shuttle vector assay in human cells. The frequency and distribution of mutations depended on the length of the linker arm. Precise targeting was observed only for linker arms of length 2 and 6, which also yielded the highest mutation frequencies (3 and 14%, respectively). **Psoralen**-TFO with longer tethers yielded mutations at multiple sites, with the maximum distance from the triplex site limited by the linker length but with the distribution within that range influenced by the propensity for **psoralen** intercalation at A:T base-pair-rich sites. Thus, gene modification can be extended beyond the site of third strand binding but with a decrease in the precision of the targeting.

L2 ANSWER 14 OF 19 MEDLINE
AN 94329445 MEDLINE
DN 94329445 PubMed ID: 8052539
TI Site-specific targeting of **psoralen** photoadducts with a triple helix-forming oligonucleotide: characterization of **psoralen** monoadduct and crosslink formation.
AU Gasparro F P; Havre P A; Olack G A; Gunther E J; Glazer P M
CS Department of Dermatology, Yale University, New Haven, CT 06520-8059.
NC ES05775 (NIEHS)
P01-AR41992 (NIAMS)
SO NUCLEIC ACIDS RESEARCH, (1994 Jul 25) 22 (14) 2845-52.
Journal code: 0411011. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 199409
ED Entered STN: 19940914
Last Updated on STN: 19940914
Entered Medline: 19940906
AB A polypurine tract in the supF gene of bacteriophage lambda (base pairs 167-176) was selected as the target for triple helix formation and targeted mutagenesis by an oligopurine (5'-AGGAAGGGGG-3') containing a chemically linked **psoralen** derivative (4'-hydroxymethyl-4,5',8-trimethylpsoralen) at its 5' terminus (psoAG10). The thymines at base pairs 166 and 167, a 5'ApT site, were targeted for photomodification. Exposure of the triple helical complex to long wavelength ultraviolet radiation led to the covalent binding of psoAG10 to the targeted region in the supF gene and to the induction of site-specific mutations. We report here experiments to characterize the photomodification of the targeted region of the supF gene in the context of triple helix formation. An electrophoretic mobility-shift assay showed that, at low radiation doses, monoadducts at base pair 166 were the major photoadducts. At higher doses the monoadducts were converted to crosslinks between base pairs 166 and 167. HPLC analysis of enzymatically hydrolyzed photoreaction mixtures was used to confirm the electrophoresis results. A strong strand preference for specific photoadduct formation was also detected.

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	28.31	28.52

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Aug 9, 2002 (20020809/UP).

=> s psoralen and vivo
0 PSORALEN
0 VIVO
L3 0 PSORALEN AND VIVO

=> file medline biosis caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	28.58

FILE 'MEDLINE' ENTERED AT 12:34:39 ON 14 AUG 2002

FILE 'BIOSIS' ENTERED AT 12:34:39 ON 14 AUG 2002
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=> s psoralen and vivo
L4 654 PSORALEN AND VIVO

=> s l4 and mutagen?
L5 61 L4 AND MUTAGEN?

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 35 DUP REM L5 (26 DUPLICATES REMOVED)

=> d 1-35 ti

L6 ANSWER 1 OF 35 MEDLINE
TI Targeted gene knockout by 2'-O-aminoethyl modified triplex forming oligonucleotides.

L6 ANSWER 2 OF 35 MEDLINE
TI In vitro RNA synthesis from exogenous dengue viral RNA templates requires long range interactions between 5'- and 3'-terminal regions that influence RNA structure.

L6 ANSWER 3 OF 35 MEDLINE DUPLICATE 1
TI Pharmacokinetic and toxicology assessment of INTERCEPT (S-59 and UVA treated) platelets.

L6 ANSWER 4 OF 35 MEDLINE DUPLICATE 2
TI Induction and excretion of ultraviolet-induced 8-oxo-2'-deoxyguanosine and thymine dimers in **vivo**: implications for PUVA.

L6 ANSWER 5 OF 35 MEDLINE DUPLICATE 3
TI Stability of DNA triplexes on shuttle vector plasmids in the replication pool in mammalian cells.

L6 ANSWER 6 OF 35 MEDLINE DUPLICATE 4
TI Unambiguous demonstration of triple-helix-directed gene modification.

L6 ANSWER 7 OF 35 MEDLINE DUPLICATE 5
TI Activation of human gamma-globin gene expression via triplex-forming oligonucleotide (TFO)-directed mutations in the gamma-globin gene 5' flanking region.

L6 ANSWER 8 OF 35 CAPLUS COPYRIGHT 2002 ACS
TI Activation of human .gamma.-globin gene expression via triplex-forming oligonucleotide (TFO)-directed mutations in the .gamma.-globin gene 5' flanking region

L6 ANSWER 9 OF 35 CAPLUS COPYRIGHT 2002 ACS
TI Targeted gene knockout mediated by triple helix forming oligonucleotides

L6 ANSWER 10 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI Binding of nuclear proteins associated with mammalian DNA repair to the mitomycin C-DNA interstrand crosslink.

L6 ANSWER 11 OF 35 MEDLINE DUPLICATE 6
TI Potassium-resistant triple helix formation and improved intracellular gene targeting by oligodeoxyribonucleotides containing 7-deazaxanthine.

L6 ANSWER 12 OF 35 MEDLINE
TI Oxyradical-mediated clastogenic plasma factors in psoriasis: increase in clastogenic activity after PUVA.

L6 ANSWER 13 OF 35 CAPLUS COPYRIGHT 2002 ACS
TI **Mutagenic** activity of substances of plant origin

L6 ANSWER 14 OF 35 CAPLUS COPYRIGHT 2002 ACS
TI Methods of inactivating leukocytes and inhibiting cytokine production in blood products

L6 ANSWER 15 OF 35 CAPLUS COPYRIGHT 2002 ACS
TI Chemically modified triplex-forming oligonucleotide for site-directed **mutagenesis**

L6 ANSWER 16 OF 35 MEDLINE DUPLICATE 7
TI Triplex-mediated, in vitro targeting of **psoralen** photoadducts within the genome of a transgenic mouse.

L6 ANSWER 17 OF 35 MEDLINE DUPLICATE 8
TI Targeted **mutagenesis** in mammalian cells mediated by intracellular triple helix formation.

L6 ANSWER 18 OF 35 MEDLINE DUPLICATE 9
TI Targeted **mutagenesis** of simian virus 40 DNA mediated by a triple helix-forming oligonucleotide.

L6 ANSWER 19 OF 35 MEDLINE
TI Inhibition of gene expression by triple helix-directed DNA cross-linking at specific sites.

L6 ANSWER 20 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI A critical review of the genotoxic potential of electric and magnetic fields.

L6 ANSWER 21 OF 35 MEDLINE DUPLICATE 10
TI The evolution of photochemotherapy with psoralens and UVA (PUVA): 2000 BC to 1992 AD.

L6 ANSWER 22 OF 35 MEDLINE DUPLICATE 11
TI The C-terminal half of UvrC protein is sufficient to reconstitute (A)BC excinuclease.

L6 ANSWER 23 OF 35 MEDLINE DUPLICATE 12
TI The role of O6-alkylguanine in cell killing and **mutagenesis** in Chinese hamster ovary cells.

L6 ANSWER 24 OF 35 MEDLINE
TI Is the induction of pyrimidine cyclobutane dimers relevant for the high cytotoxic effect of 7-methylpyrido[3,4-c]**psoralen** plus UV-A?.

L6 ANSWER 25 OF 35 MEDLINE DUPLICATE 13
TI Do oral carotenoids protect human skin against ultraviolet erythema, **psoralen** phototoxicity, and ultraviolet-induced DNA damage?.

L6 ANSWER 26 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI REPAIR DEFECTIVE MUTANTS OF ALTEROMONAS-ESPEJIANA THE HOST FOR BACTERIO PHAGE PM-2.

L6 ANSWER 27 OF 35 CAPLUS COPYRIGHT 2002 ACS
TI Peripheral blood lymphocytes as indicator cells for in **vivo** mutation in man

L6 ANSWER 28 OF 35 CAPLUS COPYRIGHT 2002 ACS
TI Photobiological activity of some water-soluble derivatives of **psoralen**

L6 ANSWER 29 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI DRUG RESISTANT LYMPHOCYTES IN MAN AS INDICATORS OF SOMATIC CELL MUTATION.

L6 ANSWER 30 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI UNUSUAL BASE PAIRING OF NEWLY SYNTHESIZED DNA IN HELA CELLS.

L6 ANSWER 31 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI IN-**VIVO MUTAGENIC** EFFECT OF 8 METHOXY **PSORALEN** AND UV LIGHT.

L6 ANSWER 32 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI **PSORALEN** DNA PHOTO REACTION CONTROLLED PRODUCTION OF MONO ADDUCTS AND DI ADDUCTS WITH NANOSECOND UV LASER PULSES.

L6 ANSWER 33 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI SISTER CHROMATID EXCHANGES IN PHOTO CHEMO THERAPY.

L6 ANSWER 34 OF 35 MEDLINE DUPLICATE 14
TI Conversion of **psoralen** DNA monoadducts in E. coli to interstrand DNA cross links by near UV light (320-360 nm): inability of angelicin to form cross links, in **vivo**.

L6 ANSWER 35 OF 35 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI 8 METHOXY **PSORALEN** AND LONGWAVE UV LIGHT PROMOTE SISTER CHROMATID EXCHANGES.

=> d 16 bib ab

L6 ANSWER 16 OF 35 MEDLINE DUPLICATE 7
AN 96226956 MEDLINE
DN 96226956 PubMed ID: 8657733
TI Triplex-mediated, in vitro targeting of **psoralen** photoadducts within the genome of a transgenic mouse.
AU Gunther E J; Havre P A; Gasparro F P; Glazer P M
CS Department of Therapeutic Radiology, Yale University School of Medicine, New Haven, CT 06520-8040, USA.
SO PHOTOCHEMISTRY AND PHOTOBIOLOGY, (1996 Feb) 63 (2) 207-12.
Journal code: 0376425. ISSN: 0031-8655.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199607
ED Entered STN: 19960808
Last Updated on STN: 19990129
Entered Medline: 19960730
AB Light-activated psoralens can covalently modify DNA and are widely used to study nucleic acid secondary structure and **mutagenesis**. Sequence specificity can be added to the photoaddition reaction by attaching the **psoralen** to an oligonucleotide designed to recognize a double-stranded DNA binding site through formation of a triple helix. We have previously used this strategy to study targeted **psoralen** modification of a triplex binding site within the bacterial supF gene carried in viral genomes. In the present work we report the targeting of **psoralen** photoadducts in vitro to a specific site in the genome of a transgenic mouse. Both 10 base and 16 base oligonucleotide-**psoralen** conjugates were capable of sequence-specific modification of genomic mouse DNA, while a truncated 8 base conjugate was not. Light activation was necessary, and a dose dependence was demonstrated for target site modification and **mutagenesis**. The 10 base conjugate rapidly found its target, with sequence-specific binding occurring after just 10 min incubation in the presence of mouse DNA. The ability to target **psoralen** photoadducts within mammalian genomes may prove useful in the study of chromatin structure and DNA repair. Moreover, this work may lead to potential in **vivo** applications of targeted **psoralen** modification.

=> d 1, 5 bib ab

L6 ANSWER 1 OF 35 MEDLINE
AN 2001453344 MEDLINE
DN 21369973 PubMed ID: 11389147
TI Targeted gene knockout by 2'-O-aminoethyl modified triplex forming oligonucleotides.
AU Puri N; Majumdar A; Cuenoud B; Natt F; Martin P; Boyd A; Miller P S; Seidman M M
CS NIA, National Institutes of Health, Baltimore, Maryland 21224, USA.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 Aug 3) 276 (31) 28991-8. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200109
ED Entered STN: 20010814
Last Updated on STN: 20010917
Entered Medline: 20010913
AB Triplex forming oligonucleotides (TFOs) are of interest because of their potential for facile gene targeting. However, the failure of TFOs to bind target sequences at physiological pH and Mg(2+) concentration has limited their biological applications. Recently, pyrimidine TFOs with 2'-O-aminoethyl (AE) substitutions were shown to have enhanced kinetics and stability of triplex formation (Cuenoud, B., Casset, F., Husken, D., Natt, F., Wolf, R. M., Altmann, K. H., Martin, P., and Moser H. E. (1998) Angew. Chem. Int. Ed. 37, 1288--1291). We have prepared **psoralen** -linked TFOs with varying amounts of the AE-modified residues, and have characterized them in biochemical assays in vitro, and in stability and HPRT gene knockout assays in **vivo**. The AE TFOs showed higher affinity for the target in vitro than a TFO with uniform 2'-OMe substitution, with relatively little loss of affinity when the assay was performed in reduced Mg(2+). Once formed they were also more stable in "physiological" buffer, with the greatest affinity and stability displayed by the TFO with all but one residue in the AE format. However, TFOs with lesser amounts of the AE modification formed the most stable triplexes in **vivo**, and showed the highest HPRT gene knockout activity. We conclude that the AE modification can enhance the biological activity of pyrimidine TFOs, but that extensive substitution is deleterious.

L6 ANSWER 5 OF 35 MEDLINE DUPLICATE 3
AN 2001105971 MEDLINE
DN 20564272 PubMed ID: 10993885
TI Stability of DNA triplexes on shuttle vector plasmids in the replication pool in mammalian cells.
AU Lin F L; Majumdar A; Klotz L C; Reszka A P; Neidle S; Seidman M M
CS Laboratory of Molecular Genetics, National Institute on Aging, National Institutes of Health, Baltimore, Maryland 21224, USA.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Dec 15) 275 (50) 39117-24. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200102
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208
AB Triple helix-forming oligonucleotides may be useful as gene-targeting reagents in **vivo**, for applications such as gene knockout. One

important property of these complexes is their often remarkable stability, as demonstrated in solution and in cells following transfection. Although encouraging, these measurements do not necessarily report triplex stability in cellular compartments that support DNA functions such as replication and **mutagenesis**. We have devised a shuttle vector plasmid assay that reports the stability of triplexes on DNA that undergoes replication and **mutagenesis**. The assay is based on plasmids with novel variant supF tRNA genes containing embedded sequences for triplex formation and **psoralen** cross-linking. Triple helix-forming oligonucleotides were linked to **psoralen** and used to form triplexes on the plasmids. At various times after introduction into cells, the **psoralen** was activated by exposure to long wave ultraviolet light (UVA). After time for replication and **mutagenesis**, progeny plasmids were recovered and the frequency of plasmids with mutations in the supF gene determined. Site-specific **mutagenesis** by **psoralen** cross-links was dependent on precise placement of the **psoralen** by the triple helix-forming oligonucleotide at the time of UVA treatment. The results indicated that both pyrimidine and purine motif triplexes were much less stable on replicated DNA than on DNA in vitro or in total transfected DNA. Incubation of cells with amidoanthraquinone-based triplex stabilizing compounds enhanced the stability of the pyrimidine triplex.